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MEETING ABSTRACT Open Access

The effect of memantine on cerebral cortex tumor necrosis factor alpha exression in a rat model of acute hyperammonemia

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Background

Literature suggests that proinflammatory mechanisms are implicated in the pathophysiology of hepatic encephalopathy. This is mainly caused by high circulating levels of ammonia (hyperammonemia-HA), due to liver failure [1,2]. In addition, NMDA receptors are excessively activated during acute hyperammonemia and thus significally contribute to the brain damage [3]. In fact, blockage of this receptor type is beneficial in experimental models of acute hyperammonemia [3,4]. The aim of this study is to assess the effect of memantine, a non-competitive NMDA receptor antagonist, on the expression of tumor necrosis factor alpha (TNF-α), a major proinflammatory cytokine, in the brain of a rat model of acute hyperammonemia.

Materials and methods

HA was induced in male Wistar rats by two consecutive ammonium acetate intraperitoneal (i.p.) injections of 12 and 8 mmol/kg respectively [2]. Another group of rats received memantine hydrochloride (20 mg/kg) 30 minutes before the first ammonium acetate injection, while control group received saline i.p. Rats were decapitated 30 minutes after the last injection and cerebral cortex TNF-α expression was determined with reverse transcription quantitative PCR.

Results

TNF-α expression in rat cerebral cortex was significantly elevated while the administration of memantine hydrochloride diminished its expression.

Conclusions

Memantine manages to compensate the induction of TNF-α, a major proinflammatory cytokine, by acute HA, in the cerebral cortex of rats. Further research is needed in order to determine if the effect of memantine may be attributed to the blockage of NMDA receptors and if it has a similar impact on the expression of other proinflammatory cytokines.

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